Pathophysiology of Lateral Patellar Dislocation

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2.1 Introduction

Patellofemoral disorders represent 20–40% of all knee problems and can be one of the most common complaints in sports related injuries. These disorders are a major cause of disability, particularly in females, and in extreme cases may contribute to termination of athlete's career and could lead to degenerative arthritic changes of the knee joint. For these reasons, disorders and in particular patellar instability often pose a diagnostic and therapeutic dilemma for the orthopedic surgeon. This dilemma implies that usually no single pathophysiology or therapeutic approach can fully explain and solve patellofemoral instability. In fact the patellofemoral joint is biomechanically one of the most complex human articulations with different anatomical components like bone shape, capsuloligament structures, and muscle that could alone or in combination be responsible for patellar instability. These factors are often present in combination in one patient, but the severity of each pathology can be different resulting in variable patterns of instability and pain that determine that each patient is almost unique; thus the characterization in a classification is a simplification of a very complex issue. Moreover the multifactoriality and variability of pathogenesis has determined in the past numerous misunderstanding. These misconceptions have been responsible for the high variety of surgical procedures proposed to treat patellofemoral instability, leading to less than completely satisfactory clinical results also related to iatrogenic cause.

Central to the development of a rational therapy for these patients is a complete and deep knowledge of the various anatomical abnormalities that can be responsible for patellofemoral instability. For a true comprehension of the influence on patellar instability by each risk factor it is fundamental to clearly understand the biomechanical rule on which the normal physiology of the patellofemoral joint is based.

The "valgus law" underlines the prevalence of the lateral structures with respect to the medial ones [23]. The lateral knee compartment of the patellofemoral joint is normally wider than the medial one. In fact the lateral condyle is larger than the medial one with an external part of the patellar groove higher, wider, and forward with respect to the medial compartment. The external patellar facet is larger in respect to the medial facet. At the capsular level is present a prevalence of the lateral retinaculum that is stronger and wider with respect to the medial one (Fig. 2.1).

The patella is the largest sesamoid bone in the body, and resides within biarticular muscles (the quadriceps and patellar tendons). The patella functions both as a lever and a pulley. As a lever, the patella magnifies the forces exerted by the quadriceps on knee extension. As a pulley, the patella redirects the quadriceps force as it undergoes normal lateral tracking during flexion.

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Fig. 2.1 A VMO dysplasia, a tightening of the lateral structure or a coordination defect between the two structures determine a stress concentration on lateral patellar facet and at the same time a maltracking with lateral patellar subluxation

Considering these anatomical features it is easy to understand how the complex and delicate equilibrium between bony, ligamentous, and capsular structures can be easily compromised altering the forces exerted on the patella, with external forces that overcome the medial forces.

So far the comprehension and treatment of patellofemoral disorders has suffered from the lack of a generally accepted classification. In fact a clear definition of anatomopathologic categories is fundamental to avoid using symptoms or objective signs to make a diagnosis.

Dejour [12] has developed a classification of patellofemoral disorders based on anatomopathologic features and on the severity of clinical findings, in order to standardize treatment. Three major groups can be identified: patellar dislocation, pain, and pain plus anatomical disorders. The first group includes three categories where instability is associated with anatomical abnormalities, while the second group includes all the patients with patellar symptoms but without anatomical alterations. This classification clearly facilitates the treatment choices, and moreover permits one to differentiate these three populations.

Instability of the patellofemoral joint is a multifactorial problem with great variability and severity of anatomical deformities that are difficult to be clearly understood and evaluated by the clinician.

Patellofemoral instability can result from soft tissue abnormalities, such as a torn static stabilizer like the medial patellofemoral ligament or a weakened dynamic stabilizer like the distal oblique portion of the vastus medialis. Generalized ligamentous laxity must also be considered as a risk factor, especially in nontraumatic instability, but this is not yet demonstrated.

The other fundamental risk factors are the osseous abnormalities such as Patella Alta, trochlea and patellar dysplasia, rotational and axial deformities of the lower limb, with alteration of Q angle.

2.2 Soft Tissue Abnormalities

Extensor muscle dysplasia is often responsible for patellar symptoms. In fact the delicate and complex muscular mechanism that controls the joint kinematics is extremely sensible to small variation.

Ficat and Hungerford [23] in the late 1970s have considered lateral patellar compression syndrome one of the major causes of patellar symptoms and instability as well as a risk for degenerative joint disease of the patellofemoral joint. The augmented tension on the lateral retinacula increases the stress on the lateral patellar facet and simultaneously predisposes to patellar malalignment and instability, but no objective data have documented this theory. Terry [55] more recently has underlined that the iliotibial band has attachments to the patellar and quadriceps tendons. Therefore excessive tension in the iliotibial band causes the patella to track in a more lateral position and predispose one to patellar dislocation.

Insall [35] and Fox [25] in the 1980s have underlined that a real hypoplasia of the vastus medialis and its altered insertion on the patella can lead to unbalanced patellar kinematics. These anatomical observations were more consistently studied in an experimental set up by Farahmand [21]. He observed in vitro that the vastus medialis obliquus has a mean orientation that deviates 47° ± 5° medially from the femoral axis, and the vastus lateralis has a mean orientation that deviates 35° ± 4° laterally from the axis [21]. He also found a different cross-sectional area between the vastus medialis and lateralis and a higher variation of this in the vastus lateralis. Therefore an imbalance in strength caused by different cross sections or different fiber orientations may lead to instability.

Vastus medialis relaxation reduces lateral patellar stability at all flexion angles. Goh [29] found lateral stability to be reduced by 30% when the vastus medialis obliquus was relaxed at 20° of knee flexion with a lateral patellar displacement of 4 mm.

A VMO dysplasia does not guarantee the force necessary to compensate the force exerted from the lateral structure to stabilize the patella in the trochlea groove. In this type of dysplasia the absence of the oblique muscle fibers causes a worse lever arm. The consequences are usually an increased patellar tilt or a tendency to patellar subluxation.

Voight [60] also has demonstrated that although the medial and lateral muscle structures are normal, a defect in the muscular coordination, can determine an opposite recruitment order between vastus medialis and lateralis originating in patellar instability.

Passive stabilizers in the patellofemoral joint include patellofemoral and patellotibial ligaments and the retinacula. Warren and Marshall describe the MPFL as an extracapsular structure [64]. The size and thickness of the ligament varies considerably among individuals, but it is relatively constant within a given person [65]. The MPFL acts as a static check rein to resist lateral translation of the patella.

Desio [16] reported that the MPFL contributes 60% of the total restraining force against lateral patellar displacement with the patellomeniscal ligament the second most important medial stabilizer contributing an average of 22% of the total restraining force. Senavongse [49] found that 20° of knee flexion was the position when 10 mm displacement occurred at the lowest restraining force. However the patella was more resistant to medial than lateral 10 mm displacement. Again Senavongse and Amis [48] tried to demonstrate the relative effects of various abnormalities on patellar stability. They found that a relaxed VMO reduced by 30% the force to displace the patella laterally in 20°–90° flexion range, while only by 14% in extension. If the MPFL was ruptured the force required to displace the patella laterally was reduced by 50% in the extended knee, decreasing while the knee flexed. Interestingly abnormal trochlear geometry reduced the lateral stability by 70% at 30° of flexion.

General hyperlaxity can also be a cause of patellofemoral instability related to the insufficiency in controlling lateral patellar displacement.

Carson and James [8], evaluating lateral patellar displacement in response to applied load at full extension, found a significantly greater lateral patellar mobility in symptomatic and hyperlaxity patient. The same observation was performed by Fithian [24] at 30° of flexion.

Nomura in 2006 [44], in a case series, showed that a hypermobile patella and generalized joint laxity were significantly important in the recurrent patellar dislocation group compared to the control group, with hypermobile patella as a predisposing factor for dislocation. Christoforakis [9] still in 2006 has shown that release of the lateral retinaculum reduces at 10° and 20° of flexion the force required to displace the patella by 20%. These findings underline the importance of medial structures like the VMO and MPFL.

2.3 Bone Abnormalities

One of the most important anatomical abnormalities originating in patellar symptoms is the trochlear dysplasia. This pathology has often been underestimated and initially considered secondary to patellar dislocation [13,15,16]. Instead intraoperative observations have confirmed that the intercondylar groove can be found completely flat or even convex [17, 18].

The normal trochlea is concave and strictly correlated to the bony contour and depth of the overlying cartilage [49, 50]. Trochlear dysplasia is defined as a groove with a proximal flat articular zone and a distal shallow zone. [15] Trochlear dysplasia was first described many years ago by Richerand [38]. This author, in 1802, described an abnormal lateral condyle in patients with recurrent patellar dislocations.

In the presence of dysplasia, the intercondylar groove may be flattened or even convex. [17, 45] This convexity presents the articular cartilage being thicker centrally than laterally and medially. [49, 50]. These findings have been confirmed from other authors [56, 61] utilizing standard x-ray and CT images. In patients with recurrent patellar dislocation Yamada et al. [68] found the convex groove to extend twice as far during flexion as in controls. In the presence of trochlear eminence, the patella has to surmount the bump during the early flexion of the knee. [13, 15] The inadequate depth of the intercondylar groove can be total or focal, when affecting only the upper part [14].

Flattening of the groove does not allow the patella to fit into the trochlea during range of motion. Imbalance

of the patellofemoral joint with risk of patellar dislocation is created by this lack of centration, especially in the first degrees of flexion that allows the lateral structure to overtake easily the medial ones. In the presence of this deformity, the stresses are prevalently distributed on the lateral facet instead of the entire groove, originating as long term arthritic degenerative changes of the joint [13, 15]. Quantitatively the convexity (bump or boss) is pathological above 3 mm or more,

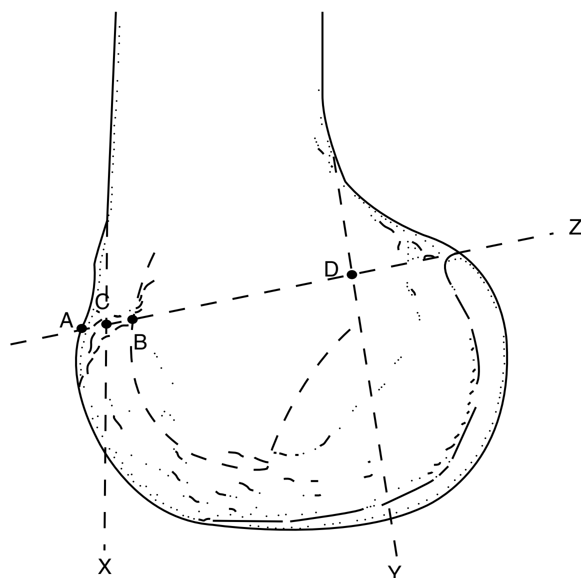


Fig. 2.2 Measurement of the trochlear boss: the distance BC (Dejour-Neyret and Gilles method; reproduced by permission from "Factors of patellar instability: an anatomic radiographic study" [15])

the depth is abnormal at 4 mm or less [15] (Fig. 2.2). The geometry of the trochlear groove has great influence on patellofemoral joint stability. The trochlear dysplasia is strongly linked with objective patellar instability, because there is a lack of congruence between the groove and the patella [15]. Recurrent patellar dislocation without surgical treatment is associated with a high incidence of the patellofemoral arthrosis [42]. There is a direct link between objective patellar instability and lateral patellofemoral osteoarthritis [11,15]. The qualitative definition of trochlear dysplasia by H. Dejour in 1990 is based on the "intersection sign" on the lateral view [14] (Fig. 2.3). D. Dejour created a classification using four grades. In Dejour and Le Countre, comparing 143 radiographs of patients and 190 control radiographs, they showed that 85% of patients with a history of patellar dislocation had evidence of trochlear dysplasia [11]. Amis et al. [2] in an in vitro study, found that trochlear dysplasia led the patella to become less stable laterally, while the trochleoplasty increased stability not significantly different from a normal knee. The importance of the lateral facet of the trochlea in resisting the lateral force is logical and widely accepted [1, 3, 48, 49]. The first author who described this concept has been Brattstrom in 1964 [7]. He studied qualitatively and quantitatively the shape of the intercondylar groove describing the trochlear dysplasia as an increase of the sulcus angle in relationship to developing defects of the trochlear profile. He found the lateral condyle to be significantly lower in patients with habitual patellar dislocation. Amis [3] in another in vitro study, showed that

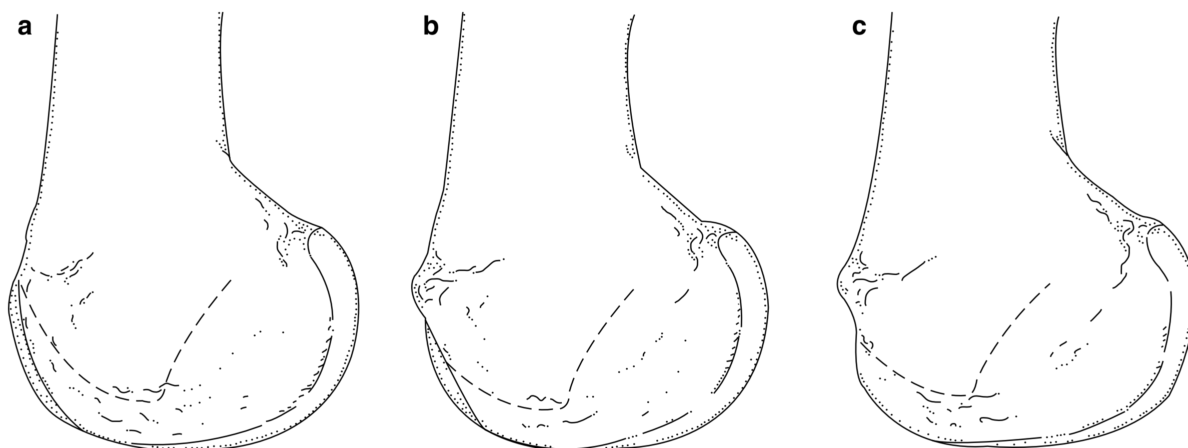


Fig. 2.3 Definition of trochlear dysplasia: the intersection of the line of the bottom of the trochlea with the two condyles allows the determination of a typology of dysplasia: (**a**) Type I; (**b**) Type II; (**c**) Type III), minor in Type I, major in Type III (Reproduced by permission from "Factors of patellar instability: an anatomic radiographic study" [15])

flattening the lateral groove had more influence on patellar laxity than dysfunction of VMO and MPFL. It has been found that the patellar shape could change in trochlear dysplasia. The distal medial facet in dysplastic knee does not articulate well with the trochlea, becoming smaller than normal. [4, 26] Fucentese et al., in a comparative MRI study, proposed that the patellar morphology may be not only a result of missing medial patellofemoral pressure in trochlear dysplastic knees, but a decreased medial patellofemoral traction. They found hypotrophic medial patellofemoral restraints and increased lateral patellar tilt in the dysplastic knees. Wiberg [67] has classified radiographically the shape of the patella determining three types of patellar hypoplasia that can originate from patellar symptoms. Ficat [22, 23] has underlined that the severe dysplasia of the internal facet implies a reduction of the weight-bearing internal area with a surface incongruence and an automatic stress concentration on lateral side that can start the degenerative phenomena.

A similar instability mechanism is encountered when Patella Alta is found. The patellar height is defined by the Insall-Salvati Ratio [37] (Fig. 2.4). Patella Alta is characterized by a more proximal position of the patella (i.e., high-riding patella). This condition has been correlated with patellofemoral dysfunction [36] and is one of the risk factors for patellar instability [39]. Dejour found in the objective patellar instability cases 24% had Patella Alta and 90% had patellar tilt [15]. High-riding patella, in fact, is strongly associated with patellar dislocation and subluxation.

Insall [36] and Blackburne [5] have underlined the role of Patella Alta as a cause of patellar instability. When the patellar tendon is longer than normal, during quadriceps contraction, the patella goes proximal and completely above the corresponding femoral surface without any lateral bony support preventing lateral dislocation. During flexion there is a delay in centration of the patella in the trochlea groove. In this condition the lateral structures do not find any bony resistance to lateral traction of the patella, due to the normal prevalence of the lateral structures with respect to the medial ones. Patella Alta, modifying the lever arm between quadriceps and patellar tendons, increases the compression forces in patellofemoral joint leading to cartilage damage. In patient with Patella Alta, Dejour has often found stiffness of the rectus femoris, supposing that Patella Alta may be a rectus femoris dysplasia [15].

Biomechanically the patellofemoral joint is a lever system. The patella is the fulcrum of this system and

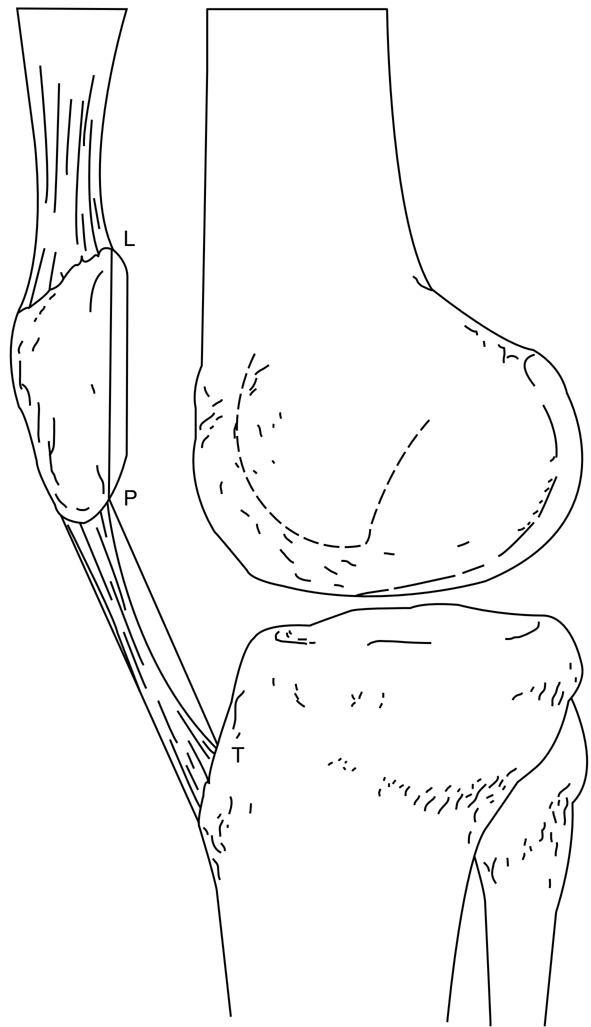


Fig. 2.4 The Insall-Salvati ratio is defined by the relation LP/LT (*LP* length of the patella, *LT* length of the patellar tendon): the normal value is between 0.8 and 1.2 (Modified and reproduced by permission from "Factors of patellar instability: an anatomic radiographic study" [15])

contributes significantly to the torque in knee extension by increasing the lever arm of the quadriceps and transmitting the forces from the quadriceps tendon to the patellar tendon. In the normal knee, the patellofemoral total contact area increases from extension to flexion and reaches a maximum at 90°, reducing the contact stress in deeper flexion. The cartilage layer is thicker in the high load area of the joint. Although in the literature some authors [33, 62, 69] have suggested that Patella Alta may altered the mechanics of knee extension, there is no consensus on the real effect of Patella Alta on

patellofemoral force, contact area, and contact pressure. Singerman, Davy, and Goldberg [51] reported, in an in vitro study, that the patellofemoral contact force, and its point of application on the patella, depended on patellar height. In a high-riding patella the magnitude of the PF contact increases with increasing flexion angle. They report also no increases from 0° to 60° of knee flexion and a significant rising at 90° in PFJ reaction force with Patella Alta. Luyckx [41], using a dynamic knee simulator, reported that the patellofemoral contact force is the sum of the patellar tendon force and the quadriceps tendon forces. In Patella Alta he showed the lowest PF contact force in initial flexion (35–70°) and a higher contact force in deeper flexion (70–120°) than in normal conditions. In this way he demonstrated a direct association between patellar height and maximal contact force. He also found that Patella Alta caused the greatest maximal contact force and pressure. In normal conditions the effective moment arm of the quadriceps tendon is greater than that of the patellar tendon because of the distal contact point of the patella during initial flexion [30, 58]. Yamaguchi and Zajac [69], moreover, by a mathematical simulation of Patella Alta to calculate a quadriceps moment arm, reported that modified lengthening of the patella or patellar tendon caused alteration of force transmission from quadriceps to patellar tendon. They showed a considerable increasing of moment arm and joint reaction force at flexion above 25–30°, with the Patella Alta condition. It seems that Patella Alta creates a more efficient knee extensor mechanism by a more distal contact point in initial flexion (0–60°), whereas, in deeper flexion, it is considered a biomechanical disadvantage [41].

Ward et al. [62, 63] demonstrated in two MRI studies that Patella Alta is correlated with a significantly larger quadriceps and smaller patellar ligament moment arm than in normal conditions, with a greater transmission force from quadriceps to patellar ligament. They showed 19% less contact area than normal between 0° and 60° of flexion, with lateral displacement and lateral tilt of the patella at 0° of flexion. Patients with Patella Alta and pain have elevated PFJ stress because of smaller PFJ contact areas and interrelate with patellofemoral cartilaginous breakdown and degeneration, dysfunction, and subsequent pain. [32, 39] No correlation could be found between malalignment and the reduced contact areas [41].

Rotational and axial deformity of the entire leg can play a role in patellar instability.

Increased femoral anteversion and/or increased tibial torsion can determine patellofemoral disorders. Smillie [52], Blaimont and Schoon [6] in the 1970s with observational studies have underlined the importance of rotational deformities in determination of patellar symptoms. Weber [66] found a frequent combination of femoral anteversion with condromalaciae and patellar instability. Eckhoff [20] and Lee [40] have demonstrated that increased femoral anteversion determined increased patellar tilt and promoted lateral patellar subluxation. Eckhoff has suggested correction of excessive femoral anteversion in young patients to prevent these phenomena. Takai [53] has documented that patients with increased femoral anteversion have an increased incidence of osteoarthritis. Femoral anteversion increases compression forces on the lateral compartment of the patellofemoral joint by bringing the lower femoral extremity in internal rotation resulting in the clinical appearance of “squinting patellae” (Fig. 2.5).

The association between distal femoral internal rotation and tibial external rotation alters the Q angle.

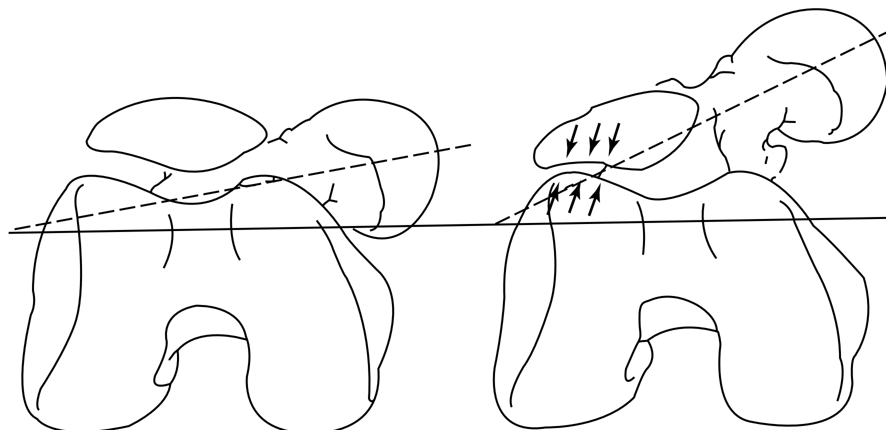


Fig. 2.5 An increased femoral anteversion increasing patellar tilt and subluxation determine higher compressive forces on external compartment of patellofemoral joint, with increased risk of chondral damage

Brattstrom [7] described the Q angle as the angle formed by the line of pull of the quadriceps and that of the patellar tendon as they intersect at the center of the patella. The Q angle is largest in extension in relation to the screw home mechanism of the knee. For this measurement to be accurate the patella should be centered on the trochlea. In males the Q angle is normally about 8–10° in females 15 plus or minus 5°. It should be noted that the relationship between the Q angle and clinical signs and symptoms has not always been consistent. A possible reason for the lack of association is related to the fact that there has been no consensus with respect to how this measurement should be taken, but more important is the fact that this measurement is taken statically, therefore, the contribution of abnormal segmental motions and muscle activation to the Q angle during dynamic activities may not be appreciated.

The Q angle is an expression of patellar kinematic that is guided by the static bony restraints and by dynamic muscle vectors. Therefore the analysis of the static deformities that can alter the patellar kinematic are better evaluated with CT scan taking into consideration femoral neck anteversion, distal femoral rotation, and tibial rotation. Patellar centration is more reliably evaluated by the measurement of TT-TG that considers femoral rotation as well as the rotation of tibial tuberosity (Fig. 2.6).

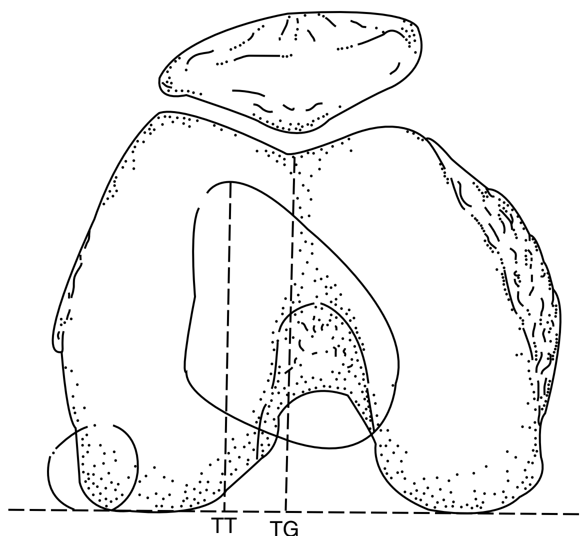


Fig. 2.6 Patellar centration evaluation with measurement of TT-TG displacement (*TT* tibial tuberosity, *TG* trochlear groove): a value greater than 20 mm is pathological (Reproduced by permission from "Factors of patellar instability: an anatomic radiographic study" [15])

Lee in 1994 [40] has demonstrated in vitro that fixed rotational deformities of the distal femur increase patellofemoral contact pressure with higher risk of joint degeneration and patellar dislocation. Powers [46] and Tennant [54] have shown that femoral internal rotation influences patellar alignment and kinematic. Powers using dynamic MRI in patient with patellar instability demonstrated that the primary contributor to patellar tilt and displacement was femoral internal rotation and not patellar motion. This phenomena was more pronounced in the last 10° of extension.

Many authors have suggested that patellofemoral symptoms are often associated with excessive primary or secondary tibial torsion [19, 25, 28]. Turner [57] has demonstrated that an excessive external tibial torsion determines a modification of the Q angle and that tibial external rotation was significantly different in patient with patellar instability. This alteration creates a less favorable lever arm for quadriceps muscle that during contraction moves the patella laterally increasing instability (Fig. 2.7).

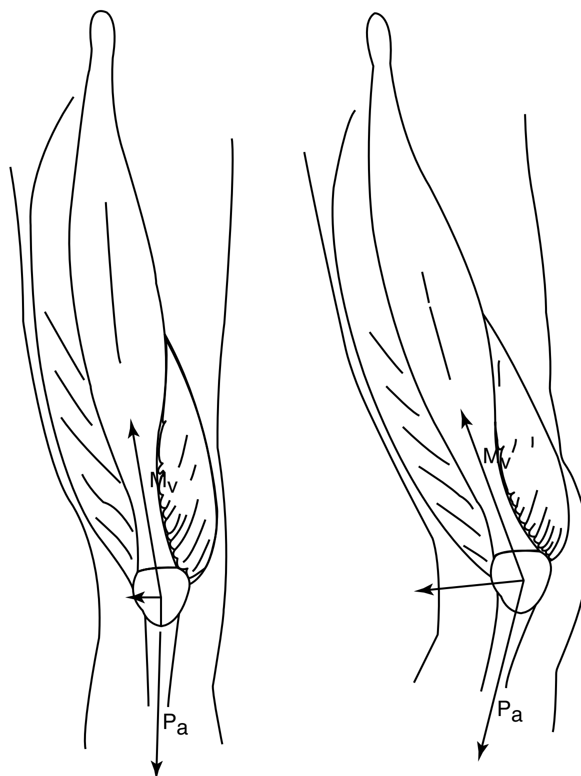


Fig. 2.7 An excessive external tibial torsion determine Q angle alteration, with a less favorable lever arm for quadriceps muscles. During contraction the forces that move the patella laterally are higher increasing instability

Van Kampen and Huiskes [59], Nagamine [43], and Sakai [47] examined the effect of tibial rotation on patellar three-dimensional movement. Hefzy [31] also studied the change of patellofemoral contact area with tibial rotation.

Apart from abnormal rotations in the transverse plane, excessive frontal plane malalignment can also influence patellofemoral joint.

Fujikawa et al. [27] observed that in varus deformity the patella displaces laterally and the lateral facet is hyperstressed with the increased risk of patellar instability. They also observed an association of proximal tibial rotation with varus deformity.

Similar combinations of varus and tibial torsion have been described by Coscia [10] in 1983. In these patients there is an increased risk of patellar instability, moreover the screw home mechanism is reduced or missed and this can originate in degenerative changes of the medial femorotibial compartment and of the lateral patellofemoral joint.

Ficat described this phenomena as a cruciate arthritis.

A valgus knee alters the Q angle and can be responsible for dynamic patellar instability. Old observational studies have underlined that an excessive valgus knee alignment associated with external tibial rotation determines especially close to extension a lateral patellar displacement especially during quadriceps contraction that increases the risk of patellar instability [22, 23].

Coscia [10] has also observed that in a valgus knee it is difficult to achieve knee extension stability due to excessive internal rotation. Therefore these knees remain unstable. During time this pathological situation leads to medial capsular distension further increasing knee laxity. In severe valgus articular stability is lacking due to the difficulties in controlling external rotation and the screw home mechanism (Fig. 2.8).

As underlined by Powers [46] a valgus knee is not only determined by static osseous abnormalities but

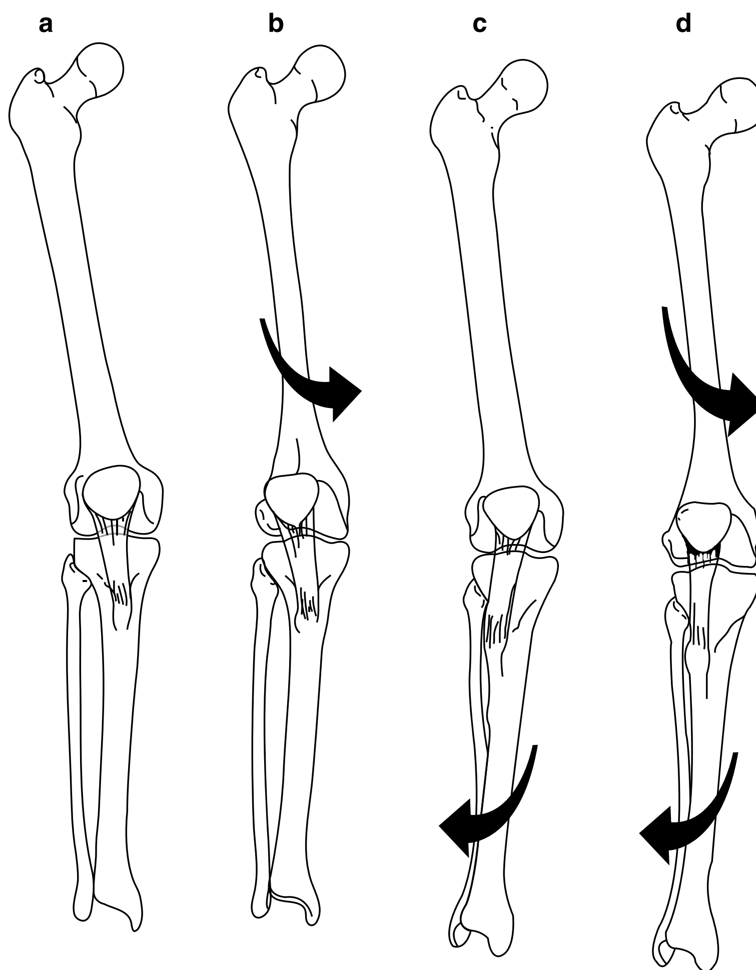


Fig. 2.8 Summary of alterations of lower limb that can originate patellar symptoms: (**a**) normal limb alignment; (**b**) increased femoral anteversion with internal rotation of femoral condyle causing higher patellar stress and instability; (**c**) external tibial torsion promotes increased compressive forces on lateral patellar facet with subluxation; (**d**) limb alterations can be combined in a same patient, with consequently a severe clinical picture and a technically demanding solution

also dynamically during certain activities as a result of femoral, tibial, or combined adduction moment. These can results from muscle weakness or imbalance, or abnormalities at the level of the hip and pelvis as well as of the foot.

Torsional defect of the lower extremity can be found often together with different patient penetration originating in a wide variety of clinical aspects that are really difficult to be globally understood.

In all these studies, a pathological value for varus/valgus or rotational deformity that is correlated to clinical symptoms has not been detected.

As we have shown the anatomical alterations that can be present with different penetration in each patient are various and complex and create several clinical aspects. Therefore the treatment options should be chosen in relation to the etiologic factors responsible for clinical symptoms in each patient.

A rational treatment of these disorders must foresee the execution of different surgical procedures in the same patient when the symptoms have a multifactorial origin in a manner to completely modify the joint physiology and kinematic.

Even if the surgical procedure acts mostly on passive and static stabilizers of the patella it is fundamental to achieve during surgery a dynamic patellar equilibrium with correct patellar tracking during the whole range of motion. Hughston [34] in 1989 has underlined the importance of dynamic stability of patellofemoral joint.

2.4 Summary

Patellofemoral instability:

- Subjective instability with anatomical abnormalities
- Traumatic dislocation without anatomical abnormalities
- Dislocation with anatomical abnormalities
- Patellofemoral pain

Patellofemoral instability:

- Soft tissue abnormalities
- Osseous abnormalities
- Soft tissue and osseous abnormalities

Soft tissue abnormalities:

- Extensor muscle dysplasia
- Ipoplasia of the vastus medialis
- Patellofemoral, patellotibial ligaments, and retinacula disorders
- General hyperlaxity

Osseous abnormalities:

- Trochlear dysplasia
- Patella Alta
- Rotational and axial deformity of lower limb
- Patellar dysplasia

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